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1st Plenary Session on 'Obesity'

Pathophysiology of obesity

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The rapidly rising prevalence of obesity, worldwide, has prompted re-evaluations of the definitions and diagnostic criteria, and of the extent of the burden it contributes to health care services. Although categorized arbitrarily for epidemiological purposes according to BMI > 25 kg/m^2 ('overweight') and BMI > 30 kg/m^2 ('obese'), the disease itself (ICD code E.66) is the process of excess fat accumulation. It leads to multiple organ-specific pathological consequences, particularly if there is a tendency to intra-abdominal fat accumulation. The simplest field method to identify obesity and risk of medical problems is the waist circumference, and this method has found a special role in health promotion. Risks begin with waist > 80 cm (women) or > 94 cm (men). As a broad generalization, obesity produces few symptoms below the age of 40 years, but then several symptoms often develop; tiredness, breathlessness, back pain, arthritis, sweatiness, poor sleeping, depression and menstrual disorders all being common. The symptoms are often attributed to diseases in other body systems. Metabolic diseases like diabetes, hyperlipidaemia and, hypertension develop later, but the mean BMI at diagnosis of diabetes is 28 kg/m^2. Ultimately, obesity increases the likelihood of myocardial infarction, stroke and several major cancers, but its biggest impact on health, especially in the elderly, is probably the multiplicity of effects on other body systems. The greatest challenge for public health is to develop effective preventive measures, recognizing that BMI > 25 kg/m^2 before the age of 20 years is a very strong predictor of obesity and ill health in adulthood.

Obesity: Cardiovascular disease: Cancer: Quality of life

The prevalence of obesity has more than doubled in the last decade, and continues to increase, giving rise to some obvious problems, and also some problems which are not so plain. The average weight of Scottish men is 72 kg at age 16–24 years, and 84 kg at 55–64 years, the corresponding values being 62 and 70 kg for women (Scottish Health Survey; Scottish Office, Department of Health, 1995). These cross-sectional data suggest that most individuals gain about 10 kg between the ages of 20 and 50 years (on average 0·3 kg/year) and this value is probably an under-estimate as younger individuals are now much fatter than they were 30 years ago. However, the rate of weight gain is much greater amongst those individuals who become obese, and is extreme in some cases. It can be assumed that this weight gain is mainly adipose tissue, which stores about 29·3 MJ (7000 kcal)/kg. About 20–30 % of 18 year olds are already overweight (BMI > 25 kg/m^2; Scottish Intercollegiate Guidelines Network, 1996), and many of them will increase over adult life to exceed a BMI of 30 kg/m^2, contributing to the 25 % of 55–64 year olds currently 'obese' in the UK. As the incidence of smoking falls, the rising level of obesity is likely to become a more dominant cause of CHD and other diseases, perhaps with different clinical characteristics from those previously seen.

What is obesity?

The word obesity (from the Latin ob-esum, meaning on account of having been eaten) is a lay term which means the same as fatness but with moderately abusive overtones. Obesity is a disease with International Classification of Disease code E66 (World Health Organization, 1997). In

Abbreviations: NIDDM, non-insulin-dependent diabetes mellitus.
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adults 'obesity' is now defined by international convention to indicate the state of having a BMI of $< 30\,kg/m^2$ (or 'grade 2 obesity'), while a BMI of $> 25\,kg/m^2$ is designated 'overweight' (or 'grade 1 obesity') and a BMI of $18{\cdot}5–25\,kg/m^2$ is 'normal', so by definition a BMI of $> 25\,kg/m^2$ is abnormal (World Health Organization, 1995). These BMI cut-offs were initially based on life-expectancy data from life assurance companies, but they match the overweight-related risks for a range of morbidities. Since the BMI (kg/m^2) is conceptually complex and inaccessible to most of the general public, an alternative measure of overweight and obesity and its health risks was required for health promotion purposes. Waist circumference is a more recently standardized alternative (measured between lowest rib and iliac crest, with the subject standing), and it relates both to total fatness and specifically to the intra-abdominal fat without the need to adjust for height (Han *et al*. 1997).

The disease itself is the process of excess fat accumulation, and obesity is a progressive systemic disease process, with multiple organ-specific manifestations. Obesity brings a host of debilitating symptoms (physical, psychological, social and medical consequences; Table 1), plus secondary metabolic effects, many of which conspire to cause IHD. Ultimately obesity can kill, through its contribution to diabetes, CHD, cancers and more directly via complications such as Pickwickian syndrome, sleep apnoea, venous thrombosis embolus, or cellulitis. Many of the pathological consequences are manifestations of the 'metabolic syndrome', which is characterized by both central fat distribution and excess total body fat. These two components are under separate genetic influences, but both are aggravated by inactivity and a high-fat diet.

Obesity and cardiovascular disease

A scan of the world literature would suggest that obesity is almost exclusively perceived by clinical scientists as a risk factor (or 'surrogate') for cardiovascular disease. Most individuals with cardiovascular disease are overweight or obese (82 % of people with cardiovascular disease in 1995 in Scotland; Scottish Office, Department of Health, 1995). To give some indication of the scale of influence of obesity on cardiovascular disease, the Scottish Health Survey (Scottish Office, Department of Health, 1995) found the prevalence of any cardiovascular disorder was 37 % of adults with BMI $> 30\,kg/m^2$ and 21 % for those with a BMI of $25–30\,kg/m^2$, compared with 10 % for individuals with BMI $< 25\,kg/m^2$. It is not possible to state from these epidemiological data which are the dominant mechanisms relating weight gain to cardiovascular disease. There are four aspects to obesity in cardiovascular disease (Table 2). First, obesity has a direct effect, causing or contributing to cardiac pathology via accelerated atheroma and also increased thrombotic risks. Second, obesity causes individuals with diseased (or even relatively normal) hearts to develop cardiac symptoms, i.e. disease as it presents. Third, obesity can mimic cardiac symptoms of breathlessness, oedema and chest pain; 40 % of obese patients with angina do not have demonstrable coronary artery disease (Bahadori *et al*. 1996). Fourth, obesity can compound and exaggerate the effect of other risk factors.

The interaction of overweight and obesity with conventional major risk factors for cardiovascular disease is very striking, as demonstrated in 12-year follow-up data from the Boston Nurses Study (Manson *et al*. 1990). In the absence of a risk factor (non-insulin-dependent diabetes mellitus (NIDDM), smoking, hypertension, hypercholesterolaemia) there was a highly significant doubling of risk as BMI increased from 21 to $> 29\,kg/m^2$ (Fig. 1). This huge effect, however, was dwarfed by the colossal rise in CHD risk when one of the risk factors was also present. There was a particularly dramatic increase in risk for smokers who were overweight (or the overweight who smoke), which argues for prioritizing smoking cessation above weight loss.

The interaction between smoking and obesity is complicated. Both factors now occur mainly in more deprived population groups, thus compounding the cardiovascular risk that arises from poor diet composition. Smoking tends to reduce appetite and to elevate metabolic rate by its thermogenic effects; so historically, therefore, smoking has been associated with thinness. Now, however, the marketing of tobacco, especially to girls, as a way of controlling weight

Table 1. Medical consequences of overweight and obesity: a systemic disease with organ-specific manifestations

Physical symptoms	Metabolic problems
Breathlessness	Hypertension
Varicose veins	NIDDM
Back pain	Hyperlipidaemia
Arthritis	Hypercoagulation
Oedema, cellulitis	IHD and stroke
Sweating	Endocrine
Stress incontinence	Hirsutism
Anaesthetic and surgical hazards	Oligomenorrhoea, infertility
Sleep apnoea	Menorrhagia
Wound dehiscence	Oestrogen-dependent cancers (breast, endometrium, prostate)
Hernia	
Chest infections	
Social	Psychological
Isolation	Tiredness
Agoraphobia	Low self esteem
Unemployment	Self deception and distortion of thought
Family, marital stress	
Discrimination	Depression

NIDDM, non-insulin-dependent diabetes.

Table 2. Four categories of mechanisms relating weight gain to cardiovascular disease

Obesity causes cardiac pathology:	Atheroma
	Thrombosis
	LV hypertrophy
	Arrhythmia
Obesity provokes cardiac symptoms:	Breathlessness
	Oedema
Obesity mimics cardiac symptoms:	Angina
Obesity compounds other risk factors:	Smoking
	NIDDM
	Hyperlipidaemia
	Hypertension

LV, left ventricular; NIDDM, non-insulin-dependent diabetes mellitus.

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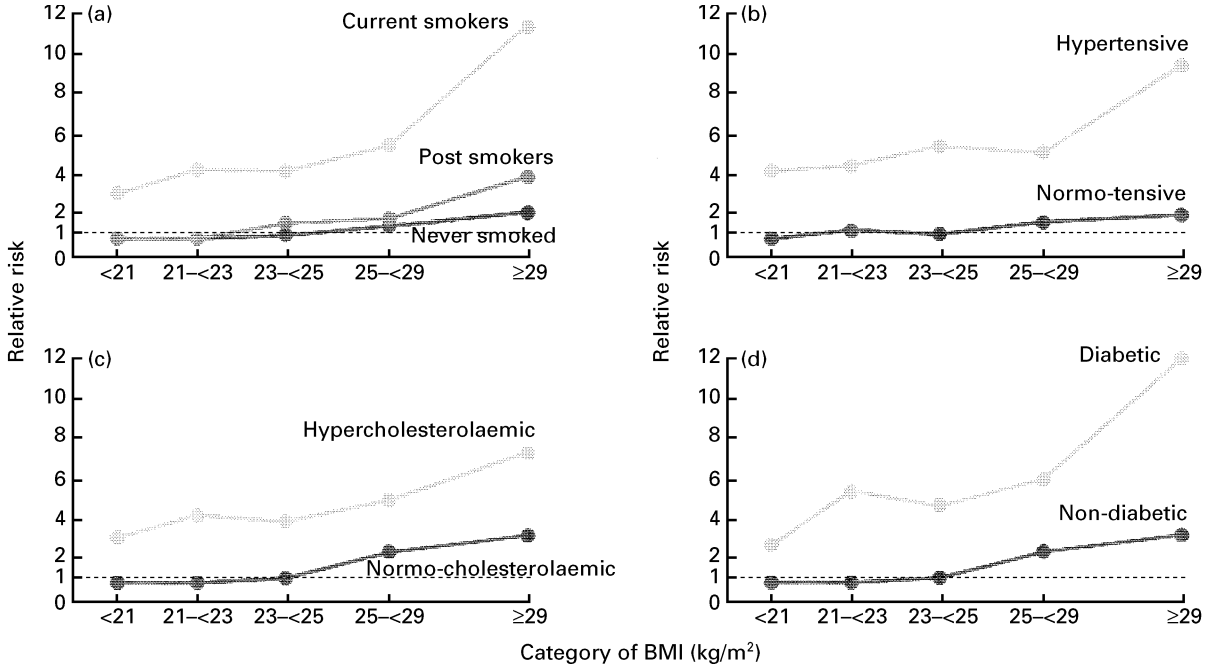

Fig. 1. Relative risks of non-fatal myocardial infarction and fatal CHD for risk factors (a) smoking, (b) hypertension, (c) hypercholesterolaemia and (d) non-insulin-dependent diabetes mellitus, with increasing BMI in the Boston Nurses Study. (From Manson *et al.* 1990.)

has resulted in smoking taking a new position as a form of weight control. A consequence is that overweight individuals commonly also smoke. Fear of weight gain is an important obstacle to smoking cessation. A recent survey in Glasgow found that 80 % of current smokers wanted to stop, and 45 % cited fear of weight gain as a barrier (J Halim, MEJ Lean and S Morris, unpublished results). The interaction between smoking and being overweight, in terms of cardiac risk, is particularly frightening (Manson *et al.* 1990). At a BMI of > 29 kg/m^2 there is a doubling of coronary risk amongst non-smokers. The risk for overweight smokers rises more steeply with BMI, to reach a 12-fold risk at BMI > 29 kg/m^2.

Obesity is undoubtedly an important primary cause of IHD and stroke, operating through the effects of weight gain on hypertension (Dyer *et al.* 1994), hyperlipidaemia (Bjorntorp, 1990; Denke *et al.* 1993, 1994), NIDDM (Colditz *et al.* 1995) and increased blood coagulability (Hankey *et al.* 1997). These metabolic effects relate specifically to the intra-abdominal fat accumulation, so although elevated BMI brings increased cardiovascular risk, at least in younger individuals, more apple-shaped ('android') fat distribution is a stronger risk factor. Central fat distribution confers risk even in thin people, but it compounds the problems of being overweight. Thus, a large waist circumference is an important indicator of cardiovascular disease risk. The term 'central obesity' is a very confused one. In the past 'waist : hip ratio' was used and this value was assumed to be an indicator of fat distribution. However, a low hip circumference (reflecting low muscle mass at least in thinner individuals) is also a risk factor for NIDDM (Seidell *et al.* 1997; Han *et al.* 1998), and waist circumference alone is a better indicator of both total fatness and central fat accumulation (Lean *et al.* 1996; Han *et al.* 1997). The waist : hip value is a more complicated and rather contrived term, without any biological basis. There is an advantage for practical applications in health promotion in using the simple waist circumference (most men at least already know their waist) and the waist circumference is virtually unaffected by height, so no correction is needed for health promotion purposes (Han *et al.* 1997). 'Action levels' for waist circumference, initially developed for Scottish Intercollegiate Guidelines Network (1996), have now been adopted by National Institutes of Health, National Heart, Lung and Blood Institute (1998; Table 3).

The prevalences of CHD and stroke, and of risk factors for CHD are shown in Table 4, according to standard cut-offs of BMI and waist circumference. For all these health problems there is a progressive rise with BMI category. The relative risks, with reference to BMI < 25 kg/m^2 are 3–9. The effect of weight gain on NIDDM is dramatic; individuals with BMI < 21 kg/m^2 virtually never get NIDDM, but the relative risk (with reference to BMI < 21 kg/m^2) over 14 years in middle-aged women is 8 at a BMI of 25 kg/m^2, 28 at a BMI of 30 kg/m^2, and 93 at a BMI of > 35 kg/m^2. A weight gain of as little as 8–10 kg doubles the risk of NIDDM, while age-adjusted relative risk is 15 with a weight gain > 20 kg (Colditz *et al.* 1995). Risks of death from CHD (and total mortality) are clearly related to quite modest weight changes in non-smoking women, with clearly increased mortality in those who gained more than 10 kg during 16 years follow up (Manson *et al.* 1995). There is a consistent trend to reduced mortality in those individuals who lose weight. The evidence is mounting gradually that

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Table 3. Measures of health risk for use in health promotion (Scottish Intercollegiate Guidelines Network, 1996; National Institutes of Health, National Heart, Lung and Blood Institute, 1998)

		Waist circumference* (cm)	BMI (kg/m^2)
Healthy or normal:	M	< 94	18·5–25
	F	< 80	
Increasing risks:	M	94–102	25–30
	F	80–88	
High risks:	M	> 102	> 30
	F	> 88	

*Waist is measured midway between the lowest rib and the iliac crest: BMI measurement requires calibrated scales and stadiometer.

Table 4. Prevalence (%) of coronary risk factors and of IHD or stroke in adults (Scottish Office, Department of Health, 1995; Lean *et al.* 1999)

BMI (kg/m^2) ….		Normal wt < 25	25–30	> 30
Cholesterol ≥ 6·5 mmol/l:	M	9·4	17·2	24·6
	F	9·4	19·1	19·8
HDL ≤ 0·9 mmol/l:	M	13·5	24·5	39·7
	F	3·4	5·8	11·8
NIDDM:	M	0·6	1·8	4·7
	F	0·4	1·0	3·8
IHD or stroke:	M	1·9	5·3	9·7
	F	1·7	3·1	7·9

NIDDM, non-insulin-dependent diabetes mellitus.

intentional weight loss reduces the mortality towards that of the general population (Lean *et al*. 1990; Williamson *et al*. 1995, 1999).

Cardiac symptoms of obesity

In general, symptoms of obesity are uncommon under age 40 years. However, several symptoms of CHD are aggravated by obesity and may be precipitated in patients who experience a rapid weight gain. These symptoms include angina and heart failure, and both are complicated. First, 40 % of obese patients with angina do not have demonstrable coronary artery disease (Bahadori *et al*. 1996), i.e. angina may be a direct symptom of obesity, with no need to blame 'cardiac syndrome X'. Second, the major symptoms of congestive heart failure are commonly mimicked or compounded by exertional and nocturnal breathlessness as a direct effect of obesity, and leg oedema secondary to venous and lymphatic obstruction at the inguinal canal in obesity. Obesity also aggravates or precipitates heart failure through an increase in left ventricular mass. This, and potentially fatal arrhythmias, are particularly found in patients with obesity-related sleep apnoea syndrome.

Obesity and cancer

There are well established links between obesity and elevated risk of several major cancers (Department of Health, 1998). Several of these (uterus, prostate, breast) are endocrine cancers, and the process appears to be mediated by elevation of free oestrogen, partly through altered adipose tissue aromatase activity, and partly through the suppression of sex hormone-binding globulin in obesity. In breast cancer, excess body fat probably delays detection of small tumours, with greater likelihood of metastases at the time of diagnosis.

There may be additional common components related to a high-fat diet. Colon cancer is also substantially more frequent in the obese. Here the mechanisms are more obscure, but physical inactivity appears to play a specific role and a high-fat diet may have specific effects. High fruit and vegetable consumption protect against colonic cancer, but it is difficult to be sure if overweight people eat less fruits and vegetables than others.

Obesity and the whole patient

Obese patients may present with one symptom, or a related set of problems that impinge on an aspect of life, but it is important to recognize that the disease produces pathology in many organs and systems concurrently. The list in Table 1 is long, but most medical consequences are probably directly, causally and reversibly related to weight gain; thus, they often develop together, gradually over the same time period. This development of secondary consequences of obesity is always slow, over many years, and the insidious debilitation may go unrecognized or ignored. From the clinical aspect, therefore, it is important to undertake a full evaluation of patients, and to enquire about other systems which may still be affected, but sub- clinically. In that way it may be possible to check the progression of a range of problems.

There are no detailed longitudinal surveys available to examine the full complexity of obesity symptomatology. Virtually all the symptoms and consequence of obesity are multifactorial and also age-related. Thus, establishing thresholds is difficult, and particularly so because body fat and BMI commonly increase with age. As a very broad generalization, direct specific 'medical' symptoms of obesity are rare below the age of 40 years, and then tend to appear in clusters. It is apparent, however, that pathological consequences start at much younger ages; therefore, effects such as elevated blood pressure, hyperlipidaemias or insulin resistance can be detected in young individuals. Endocrine consequences, such as infertility and polycystic ovarian syndrome present in obese young women. Hazards of obesity in pregnancy are well documented, including metabolic disturbances such as gestational diabetes, or a striking increase in pre-eclampsia in overweight women (Sattar *et al*. 1996), and increased likelihood of mechanical difficulties during labour. The commonest major symptom of obesity is probably breathlessness, often attributed to asthma and treated ineffectively with bronchodilators. The progression to sleep apnoea syndrome and Pickwickian syndrome is one of the least understood hazards. It is most common with

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central fat distribution and has mechanical as well as central (medullary) components. An early warning system is snoring, and this condition can cause death, both through respiratory failure and through road accidents which result from falling asleep while driving.

Operative and post-operative surgical complications are more frequent in obese subjects of all ages, particularly chest infections, wound infections and venous thrombosis which seems resistant to prophylaxis. With increasing age, obesity is the strongest predictor for venous thrombosis (Goldhaber *et al*. 1983; Lowe *et al*. 1999). An interesting new finding in this context is the increased adipose tissue synthesis of plasminogen activator inhibitor in the obese (Bastard & Pieroni, 1999).

The highly-publicised relationship between obesity and cerebrovascular and cardiovascular diseases (stroke, myocardial infarction) is very real, but related to age in a complicated way. Prospective studies hitherto have not been able to delineate clearly whether relative risks of coronary risk factors change with age. Absolute risk increases dramatically, but there is some suggestion that relative risk is little changed with age (Lean *et al*. 1998). On the other hand, the evidence from other large epidemiological studies suggests that the impact of obesity on BMI or CHD declines with age. The prospect of accelerated CHD is a major worry for young men, but the added risk from obesity appears to be lost by the age of years 50 years (Royal College of Physicians, 1983). For older individuals, therefore, the burden of ill health from obesity is dominated by musculo-skeletal, metabolic and socio-psychological problems, and CHD is less important as a result of obesity.

Psychological disturbances are common amongst obese and overweight individuals, and even in normal-weight individuals who are 'restrained eaters' in order to control an underlying genetic predisposition to weight gain. It is considered that the disturbances of thought, and beliefs about body image and the systematic under-reporting of food intake are all secondary, rather than driving the hyper-phagia of obesity. The declared depression of the overweight is a very real problem, but with rather different characteristics than the classical 'endogenous depression'. First, it is associated with overeating, often in a planned but surreptitious bingeing pattern, whereas endogenous depression is characterized by weight loss. Second, it appears to resolve after major weight loss (Sjostrom *et al*. 1999). Binge eating in obesity is frequent, but little under-stood. It is itself probably a secondary problem, but may in fact underlie at least the maintenance of obesity. Regulating eating patterns by behavioural (or surgical) means can reverse the problem.

Conclusion

Over history, there have been times when obesity has been treated as a mark of success or stature in society, as an object of humour, and as a sensitive personal problem. Somewhat grudgingly, it seems, obesity has become accepted as a risk factor for CHD, with a major influence on other risk factors. However, just listening to patients makes it clear that obesity is a major disease, the debilitating consequences of a relentless disease process. It certainly does cause heart disease, but this disease is only one of many consequences, and a rather distant one. The reality of life with obesity is steady accumulation of problems affecting every aspect of life and almost every system of the body.

Various attempts to estimate the economic costs of obesity have produced misleading information, because data relating BMI category and age to costable disease consequences are very incomplete, and some relative risk values are very misleading. The value calculated by Seidell (1995) of 4 % of the total health care budget attributable to BMI $> 25\,kg/m^2$ (i.e. above the costs if everyone had BMI $< 25\,kg/m^2$) is enormous, but possibly conservative. Also, as the prevalence of overweight and obesity rise, not only will these costs rise, but the whole profile of some clinical practice will change. For example, as smoking declines, heart disease will fall, but the pathological and clinical pattern of CHD will also change as obesity becomes responsible for a greater proportion of CHD.

The conventional BMI and waist cutoffs (Table 3) give good indicators of multiple health risks, independent of age. They can form the basis for epidemiology, health promotion and clinical screening, although interpretations will vary to some extent between racial and ethnic groups.

References

Bahadori B, Neuer E, Schumacher M, Fruhwald F, Eber B, Klein W, Toplak H & Wascher TC (1996) Prevalence of coronary-artery disease in obese versus lean men with angina-pectoris and positive exercise stress test. *American Journal of Cardiology* **77**, 1000–1001.

Bjorntrop P (1990) Obesity and adipose tissue distribution as risk factors for the development of disease – a review. *Infusion-therapie* **17**, 24–27.

Bastard JP & Pieroni L (1999) Plasma plasminogen activator inhibitor 1, insulin resistance and android obesity. *Biomedicine and Pharmacotherapy* **53**, 455–461.

Colditz GA, Willett WC, Rotnitzky A & Manson JE (1995) Weight gain as a risk factor for clinical diabetes mellitus in women. *Annals of Internal Medicine* **122**, 481–486.

Denke MA, Sempos CT & Grundy SM (1993) Excess body weight. An under recognised contributor to high blood cholesterol levels in white American men. *Archives of Internal Medicine* **153**, 1093–1103.

Denke MA, Sempos CT & Grundy SM (1994) Excess body weight. An under recognised contributor to dyslipidaemia in white American women. *Archives of Internal Medicine* **154**, 401–410.

Department of Health (1998) *Nutritional aspects of the development of cancer. Report of the working group on diet and cancer of the Committee on Medical Aspects of Food and Nutrition Policy (COMA)*. London: H.M. Stationery Office.

Dyer AR, Elliott P, Shipley M, Stamler R & Stamler J (1994) Body mass index and associations of sodium and potassium with blood pressure in INTERSALT. *Hypertension* **23**, 729–736.

Goldhaber SZ, Savage DD, Garrison RJ, Castelli WP, Kannel WB, McNamara PM, Gherardi C & Feinleib M (1983) Risk factors for pulmonary embolism. The Framingham Study. *American Journal of Medicine* **74**, 1023–1028.

Han TS, Feskens EJM, Lean MEJ & Seidell JC (1998) Associations of body composition with non-insulin dependent diabetes mellitus. *Diabetic Medicine* **15**, 129–135.

Han TS, Seidell JC, Currall JEP, Morrison CE, Deurenberg P, Lean MEJ (1997) The influences of height and age on waist

https://doi.org/10.1017/S0029665100000379 Published online by Cambridge University Press

circumference as an index of adiposity in adults. *International Journal of Medicine* **21**, 83–89.
Hankey CR, Rumley A, Lowe GDO, Woodward M & Lean MEJ (1997) Moderate weight reduction improves red cell aggregation and factor VII activity in overweight subjects. *International Journal of Obesity* **21**, 644–650.
Lean MEJ, Han TS & Deurenberg P (1996) Predicting body composition by densitometry from simple anthropometric measurements. *American Journal of Clinical Nutrition* **63**, 4–14.
Lean MEJ, Han TS & Seidell JC (1998) Impairment of health and quality of life in people with large waist circumference. *Lancet* **351**, 853–856.
Lean MEJ, Han TS & Seidell JC (1999) Impairment of health and quality of life using new US Federal guidelines for the identification of obesity. *Archives of Internal Medicine* **159**, 837–843.
Lean MEJ, Powrie JK, Anderson AS & Garthwaite PH (1990) Obesity, weight loss and prognosis in type 2 diabetes. *Diabetic Medicine* **7**, 228–233.
Lowe GDO, Haverkate F, Thompson SG, Turner RM, Bertina RM, Turpie AGG & Mannucci PM on behalf of the ECAT DVT Study Group (1999) Prediction of deep vein thrombosis after elective hip replacement surgery by preoperative clinical and haemostatic variables: The ECAT DVT Study. *Thrombosis and Haemostasis* **81**, 879–886.
Manson JE, Colditz GA, Meir J, Stampfer MJ, Willett WC, Rosner B, Monson RC, Speizer FE & Hennekens CH (1990) A prospective study of obesity and risk of coronary heart disease in women. *New England Journal of Medicine* **322**, 882–889.
Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, Hennekens CH & Speizer FE (1995) Body weight and mortality among women. *New England Journal of Medicine* **333**, 677–685.
National Institutes of Health, National Heart, Lung and Blood Institute (1998) Clinical Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults – The Evidence Report. http://www.nhlbi.nih.govguidelines/obesity/ob home.htm
Royal College of Physicians (1983) Obesity. A Report of the Royal College of Physicians. *Journal of the Royal College of Physicians London* **17**, 5–65.
Sattar N, Gaw A, Packard CJ & Greer IA (1996) Potential pathogenic roles of aberrant lipoprotein and fatty acid metabolism in pre-eclampsia. *British Journal of Obstetrics and Gynaecology* **103**, 614–620.
Scottish Intercollegiate Guidelines Network (1996) Obesity in Scotland: Integrating Prevention with Weight Management. http://www.show.scot.nhs.uk/sign/sign8inf.htm
Scottish Office, Department of Health (1995) Scottish Health Survey 1995 [W Dong and B Erens, editors]. Edinburgh: The Stationery Office.
Seidell JC (1995) The impact of obesity on health status: some implications for health care costs. *International Journal of Obesity* **19**, S13–S16.
Seidell JC, Han TS, Feskens EJM & Lean MEJ (1997) Narrow hips and broad waist circumferences independently contribute to increased risk of NIDDM. *Journal of Internal Medicine* **42**, 401–406.
Sjostrom CD, Lissner L, Wedel H & Sjostrom L (1999) Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS Intervention Study. *Obesity Research* **7**, 477–484.
Williamson DF, Pamuk E, Thun M, Flanders D, Byers T & Heath C (1995) Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40–64 years. *American Journal of Epidemiology* **141**, 1128–1141.
Williamson DF, Pamuk E, Thun M, Flanders D, Byers T & Heath C (1999) Prospective study of intentional weight loss and mortality in overweight white men aged 40–64 years. *American Journal of Epidemiology* **149**, 491–503.
World Health Organization (1995) *Physical Status: The Use and Interpretation of Anthropometry. WHO Technical Report*. Geneva: WHO.
World Health Organization (1997) *International Statistical Classification of Disease and Related Health Problems*, 10th ed. Geneva: WHO.

https://doi.org/10.1017/S0029665100000379 Published online by Cambridge University Press